The diversity of maternal-age effects upon pre-adult survival across animal species

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Edward Ivimey-Cook[1*] and Jacob Moorad[1]

[1]Institute of Evolutionary Biology, School of Biological Sciences, University of Edinburgh, Edinburgh, EH9 3JT, UK

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Diversity of maternal-age effects

Corresponding author

E.Ivimey-Cook@uea.ac.uk

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Abstract

Maternal senescence is the detrimental effect of increased maternal age on offspring performance. Despite much recent interest given to describing this phenomenon, its distribution across animal species is poorly understood. A review of the published literature finds that maternal age affects pre-adult survival in 252 of 272 populations (93%) representing 97 animal species. Age effects tended to be deleterious in invertebrates and mammals, including humans, confirming the presence of senescence. However, bird species were a conspicuous exception, as pre-adult survival tended to increase with maternal age in surveyed populations. In all groups, maternal-age effects became more negative in older mothers. Invertebrates senesced faster than vertebrates, and humans aged faster than non-human mammals. Within invertebrates, Lepidopterans demonstrated the most extreme rates of maternal-effect senescence. Among the surveyed studies, phylogeny, life history, and environment (e.g., laboratory vs wild populations) were tightly associated; this made it difficult to make confident inferences regarding the causes of diversity for the phenomenon. However, we provide some testable suggestions, and we observe that some differences appear to be consistent with predictions from evolutionary theory. We discuss how future work may help clarify ultimate and proximate causes for this diversity.

Introduction

Senescence is the age-related physiological deterioration of organismal function typically associated with increasing mortality risk (actuarial senescence) and decreasing fertility (reproductive senescence). Studies report actuarial and reproductive senescence in most animal species across most phyla [1–6], with especially well documented senescent declines in wild vertebrates [7–10] and laboratory invertebrates [11–14]. Maternal senescence, the detrimental result of a mother's increasing age on traits associated with offsprings' life history or fitness [15–19], is a distinctly different manifestation of age. Whilst such effects of maternal age are attracting increased scientific attention, their distributions across the tree-of-life remain poorly described [20]. Investigating the prevalence and degree of maternal-age effects is an important first step to understanding ultimate and proximate causes of this form of senescence, as this may identify taxa that have unusual manifestations of ageing that warrant special focus in the future. To clarify, we use the term 'ageing' to refer to any age-related change, but we reserve 'senescence' to indicate a deleterious effect of increased age.

Several hundreds of models have been proposed to explain the proximate causes of senescence [21–25]. In contrast, there are few explanatory evolutionary models, but all share the central tenet that senescence is caused ultimately by age-related declines in the efficacy of natural selection [26]. Mutation accumulation [27] and antagonistic pleiotropy [28] are two such models that make different assumptions regarding the genetic architecture of age-specific traits. Population genetic models use estimates of vital rates (age-specific survival and reproduction rates) and various assumptions related to gene action to predict patterns of actuarial senescence (e.g. [29,30]). More recently, Moorad and Nussey [31] modified these to quantify how age changes the strength of selection for age-specific maternal effects and to show how these changes cause maternal senescence

72 manifested upon pre-adult survival to evolve. They predicted that evolved demographic
73 patterns of this senescence should be qualitatively different from actuarial and
74 reproductive senescence. However, we know little about how well this model predicts
75 patterns of ageing in real populations.

76 In this paper, we address conspicuous gaps in our understanding of the taxonomic
77 breadth and intensity of maternal-effect ageing by performing an extensive systematic
78 review of the literature using meta-analytical methodology. We chose pre-adult survival,
79 defined here as survival throughout some part of the pre-reproductive period. The nature
80 of this part will vary according to the methodologies of the available papers, and it largely
81 reflects the characteristics of the study species (e.g., hatching rate in invertebrates,
82 survival to fledging in birds, survival to weaning in mammals or child survival in
83 humans). This trait was chosen as our focus for maternal-age effects for several reasons:
84 1) this trait's relationship to fitness is profound and well-understood conceptually [26];
85 2) evolutionary theory explicitly models age-specific maternal effects on this trait [31];
86 and 3) associations between the trait and maternal age are observed with sufficient
87 frequency to enable a large-scale review. This study addresses questions about the nature
88 of maternal-effect ageing as it manifests on pre-adult survival rates:

89 1. *Does maternal age tend to affect pre-adult survival in most species?*
90 2. *Do effects of increased maternal age tend to be negative (is maternal senescence the*
91 *norm)?*
92 3. *What features of specific studies (for example, phylogeny, presence/absence of*
93 *biparental care, nature of human interventions) appear to predict effect sizes?*

94 We find that maternal-age effects are widespread across studies of animal species.
95 However, senescence appears to be a general and important phenomenon in only some
96 groups, with large observable variation in the rates of senescence across groups. Wild

birds and Lepidopterans (butterflies and moths) represent two disparate extremes. Why these particular taxonomic groups should be unusual in this respect is an ecological and evolutionary puzzle.

Materials and Methods

This meta-analysis followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses ("PRISMA") guidelines [32] (Fig. S1). A literature search was conducted in December 2019 using the online databases Web of Science and Scopus. Search terms are provided in Supplementary Table S1.

Accepted papers include the number of surviving and dying pre-adults as functions of maternal age (Fig. S1). Where a study was replicated within a paper or where a species was studied in more than one paper, discrete binomial datasets were extracted for each replicate population, and we treated all such within-species replicates as independent in subsequent analyses. Our criteria for acceptance and methods for data extraction are described in the Supplemental Methods. Each age class was associated with a corresponding number of surviving and dying pre-adults (coded with 1s and 0s, respectively) reconstructed from the realised maternal age distribution, the mean rates of age-specific fecundity, and pre-adult survival rates extracted from the source papers.

Maternal ages in each study were standardized by dividing by generation time T. For each replicate i, generation time T_i was calculated as the average of the maternal age distribution $f(x)$, or $T_i = \sum_x xN_{xi} / \sum_x N_{xi}$ [33]. However, this measure is sensitive to the age structure and vital rates of the population. In populations where the timing of breeding is influenced by experimenters who may have wished to enhance the power of a study to detect age-related effects, the value of T in the experimental population may not reflect the distribution of maternal ages found in natural or equilibrium populations.

Such cases likely involve the over-sampling of older ages; this would tend to inflate values of T and therefore underestimate the true magnitudes of maternal effects. Laboratory studies are most likely to introduce such biases. Failure to properly incorporate the duration of the pre-reproductive period into generation time calculations, which is often substantial in laboratory invertebrates, can also contribute to significant bias. Therefore, for studies where age was defined in terms of elapsed time since reaching adulthood, total age x was taken as the sum of adult age and the duration of the juvenile period. The latter was taken either from the source paper or from secondary sources.

All else equal, we expect that the rates of survival over some time interval will decrease as the size of that interval increases. Wherever possible, we extracted estimates of this pre-adult study interval, t, from each study. This study interval is standardized by generation length. For example, survival to one year of age was assessed in the olive baboon (*Papio anubis)*, which has a generation time of 11.16 years (see [34] for estimates of T and pre-adult study duration). Here, t = 1 yr / 11.16 yr = 0.09. Some studies did not provide clear descriptions of study intervals. For example, time-to-hatch in *Drosophila melanogaster* eggs was unspecified in [35]. In cases such as these, an approximate study interval for that species was obtained from available secondary sources (e.g., 13.50 hours for *D. melanogaster* [36]).

Each study was assigned to one of four environmental groups: wild, laboratory, semi-captive (where humans provided food or veterinary intervention), and agricultural. Humans were also surveyed but not so categorized. Non-human animals were grouped by taxonomic relationships into invertebrates, birds, non-human mammals, and other vertebrates. Invertebrates were further divided into Lepidopterans and other invertebrates (including non-Lepidopteran insects), but this division was made after effect sizes were estimated, and a pronounced difference was observed between these

two groups. Phylogenetic trees were created using the National Centre for Biotechnology Information Taxonomy database [37] and PhyloT [38] and visualised using 'ggplot2' and 'ggtree' [39,40].

We estimated the effect that maternal age had on the proportion of surviving pre-adults for each replicate independently. We fit generalised linear models (GLMs) of pre-adult survival (P) with binomial error (e) distribution and probit link functions to: [1] age-independent, [2] linear and [3] quadratic models of maternal age. This link function assumes a Gaussian distribution of a latent predictor variable, and it is a standard function used by quantitative geneticists for scaling genetic contributions to survival [41,42]. For equations 1-3, $P(x)$ is the probability of a pre-adult surviving at standardised maternal age x, whereas A, B, and C are the intercept, the linear coefficient, and the quadratic coefficient, respectively.

$$P(x) = A + e \quad [1]$$

$$P(x) = A + Bx + e \quad [2]$$

$$P(x) = A + Bx + Cx^2 + e \quad [3]$$

Replicate-specific log-likelihoods for all models were noted along with estimates of effect sizes and associated standard errors (SEs). We calculated Akaike Information Criterion values (AIC) for each replicate i, and model j using $AIC_{ij} = 2k_j - 2loglik_i$, where k_j is the number of parameters (one, two or three, for the age-independent, linear or quadratic models, respectively). From these, sample-size corrected AIC values ($AICc$) were calculated using the formula $AICc_{ij} = \frac{AIC_{ij}+2k_j(k_j+1)}{(n_i-k_j-1)}$, where n_i was the number of observations for each replicate [43]. Based upon the results from model-fitting (see

171 Results), our subsequent analyses focus upon the linear effects of maternal age in old
172 mothers, where "old" is defined as equal or greater than the average age of mothers T
173 (standardized maternal ages greater than one).

174 To correct for variation in study intervals t across studies, we first assessed its impact
175 on maternal-age effects. We performed a bootstrap weighted regression using the "boot"
176 package in R Version 3.6.0 [44–46] of estimated linear effect sizes upon t. Weights were
177 taken from the inverse of the estimated SEs that were associated with the linear effect
178 sizes. Values of t are mathematically constrained on the interval $1 \geq t > 0$, and they ranged
179 here from 0.007 in a human population to 0.935 in a Dermestid beetle species,
180 *Trogoderma inclusum* (See Fig. S2 for a full distribution of t across all extracted
181 populations). In general, invertebrates appeared to spend a greater fraction of their
182 generation time observed as pre-adults than the vertebrates (median t = 0.120 and 0.091,
183 respectively). Note that two replicates of rotifers (*Brachionus calcyflorus*) included a
184 diapause phase as part of the pre-adult period [47]. As this period of diapause described
185 in the paper appeared to us to be both arbitrary and highly influential to calculated values
186 of t, we considered only the time from egg to hatching and omitted the diapause phase
187 from analysis of these populations.

188 The overall effect of t (pre-adult study interval) on maternal-age effects tended to be
189 negative, -1.235 (-2.750, 0.107) [results presented in this way express the bootstrapped
190 mean and the bias-corrected 95%-tiles in brackets]. Because greater values of t indicate
191 greater exposure to mortality risk, and some risk can follow from maternal age, we expect
192 that any average effect that age tends to have on survival across all studies will be
193 amplified as t increase. As these age effects tended to be negative (see Result below), the
194 negative influence of t upon those estimates is to be expected. We corrected age effects
195 for the estimated effect of study interval by adding $1.235 \times$ t to each. We did not alter SEs

196 associated with these estimates. Replicates were pooled in groups in accordance to
197 taxonomy and environments. From these groups, we calculated weighted bootstrapped
198 means for each (n = 10,000 replicates), where weightings were the inverse of the
199 estimated SEs.
200

201 ## Results

202 A total of 196 animal papers met our search criteria. From these, we extracted and
203 analysed 273 populations from 97 animal species (Table S2). One population was
204 disregarded because no offspring died within the experimental replicate. Species were
205 studied in a single environmental context, with three exceptions: 1) the red-legged
206 partridge, *Alectoris rufa*, (semi-captive and agricultural [48,49]); 2) the domestic sheep,
207 *Ovis aries*, (agricultural and natural [50,51]); and 3) the Columbian ground squirrel
208 *Spermophilus columbianus* (semi-captive and natural [52,53]).
209 There were strong associations among the available studies between taxonomic and
210 environmental groups (Table 1). Invertebrate species were only studied in the
211 laboratory. Birds and mammals were most frequently studied in the wild, and only one
212 study from one species from either of these groups (*Mus musculus*) provided suitable
213 laboratory data. Ideally, taxonomic groups would have been distributed more evenly over
214 environments, as this might have supported a two-factor analysis. Unfortunately, these
215 strong associations among the available literature undermine rigorous attempts to assign
216 causes of potential effect size differences to either phylogeny (at the
217 vertebrate/invertebrate scale) or environmental context. Compounding this problem is
218 the fact that life history strategies share this same dichotomous partition of species: birds
219 and mammals are long-lived and tend to provide obvious post-natal maternal care. In

220 contrast, the invertebrate species studied here in the laboratory are short-lived and
221 demonstrate little or no conspicuous maternal care. The sole mammalian species to be
222 studied in the laboratory is the only species studied in that context to provide post-natal
223 maternal care. The only studied reptile species, *Lacerta vivipara*, provides pre-natal
224 maternal care.

225 Replicate-specific results from the GLMs (Equations 1-3) are given in Table S3a.
226 Comparisons of AICc values found that the age-independent models were best in 20
227 cases, linear age effect models were best in 51 cases, and quadratic age effect models
228 were best in 201 cases. Summed AICc values over all replicates indicated a strong
229 preference for the quadratic model of maternal age on pre-adult survival (ΔAICc Age-
230 Independent: +229755; ΔAICc Linear: +43828). We estimated negative quadratic effects
231 in 202 cases and positive quadratic effects in 70 cases. The weighted bootstrapped means
232 of the quadratic effects were negative when pooled over all species -0.525 (-0.813, -
233 0.308) and within all taxonomic groups (Table 1). Quadratic effects were different from
234 zero in the aggregate and in all but two groups (birds and mammals). Nevertheless, the
235 strong tendency towards a negative quadratic effect of age across species indicates that
236 linear fits of all available maternal ages tend to underestimate senescence experienced
237 by older females (or overestimate maternal effect improvement in the old). In light of this
238 finding, we re-focused our question to evaluate the linear effects of maternal age on old
239 females only, where old defines all ages greater than T (generation time). The distribution
240 of age-effects in old mothers is illustrated in Fig. 1, and the range of ages that remained
241 for each study after removing ages less than T are illustrated in Fig. S3. Note that the
242 approach taken here to focus upon particular ages does not presume that senescence
243 actually exists in any population; in this way it differs from other approaches that

estimate an age-of-onset for senescence and use this age to define ageing rates in identified senescence phases of life [54–56].

When averaged over all populations, the linear effects of maternal ages across all available ages were negative, -0.378 (-0.573, -0.204). The linear effects of ages *in the old*, which are the focus of our analyses, were stronger and remained statistically significant, -0.691 (-0.913, -0.505). All effect sizes are reported on the probit scale. Conversion to the scale of survival is not straightforward, as linearity on the probit scale implies strong nonlinearity on the scale of survival. Nonetheless, we provide one metric that can indicate the effects of increased maternal age at the onset of old age on this scale. We define $\delta(0.01 \times \mathrm{k})$ to be the increase in age from T that delivers a 1% change in pre-adult survival, where k is +1 (a 1% increase in survival) when age effects are positive ($B > 0$) and k = -1 (a 1% decrease in survival) with maternal senescence ($B < 0$). Smaller values indicate less time required to make that change and stronger age effects. These values are estimated for all replicates and reported in Table S3b and Fig. S4 of the supplementary material. The reported taxonomic structure is based upon probit measures, but the diversity illustrated in Fig S4 suggests that these qualitative patterns are robust to this change in scaling.

Populations were pooled within each environment-by-taxon group, and the bootstrapped means and 95%-tiles are reported for each in Table 1. Of the five most populated groups, all appeared to have mean effect sizes that differed from zero. Lepidopterans, other invertebrates, wild mammals, and humans exhibited statistically significant deleterious effects of maternal age. In contrast, wild birds appeared to present positive age effects on early survival. Senescence was most pronounced within the Lepidopterans, with deleterious age effects in the old of nearly an order of magnitude greater than the global mean (-6.142 vs -0.691).

Table 1. Maternal-age effects in the old for all environment-by-taxon groups (means and bias-corrected 95%-tiles). Sample sizes are given in italics where the number of species is followed by the number of replicates. Confidence intervals are not indicated when only one replicate was available. Bold-faced estimates of the means indicate significance at $\alpha < 0.05$.

	Lepidopterans	*Other Invertebrates*	*Birds*	*Mammals*	*Other Vertebrates*	*Humans*
Laboratory	**-6.142** (-8.885, -4.088) *15/27*	**-0.849** (-1.295, -0.471) *34/79*	-	3.280 *1/1*	0.075 *1/1*	-
Semi-Captive		-	-0.515 (-0.937, 0.029) *4/4*	-0.228 (-0.723, 0.045) *7/8*	-0.986 *1/1*	-
Agricultural		-	**0.327** (0.071, 1.246) *2/8*	0.137 (-0.258, 0.583) *3/13*	-	-
Wild		-	**0.124** (0.002, 0.287) *20/37*	**-0.295** (-0.451, -0.140) *11/12*	-	-
Humans		**-**	-	-	-	**-0.819** (-1.113, -0.423) *1/80*

Finally, we paired four groups and assemblages of groups to compare effect sizes using Mann-Whitney U Tests. Two such assemblages are 'Non-Human Mammals' (n = 34) and All Vertebrates' (n = 165). The bootstrapped means values for these are 0.0001 (-0.218, 0.359) and -0.303 (-0.456, -0.164), respectively. Non-directional effects within the 'Non-Human Mammals' group is caused by combining positive effects from agricultural studies with negative effects from other mammal populations. The following comparisons of mean effect sizes follow from Table 1 and results from four independent Mann-Whitney Tests:

1. Lepidopterans < Non-Lepidopteran Invertebrates < 0 (W = 198, P <0.001);
2. Humans < Non-Human Mammals (W = 532, P <0.001);

3. Wild Mammals < 0 < Wild Birds ($W = 64$, $P < 0.001$); and
4. Non-Lepidopteran Invertebrates < All Vertebrates < 0 ($W = 5501$, $P = 0.049$).

Discussion

Our results provide definitive answers to two study goals. First, maternal age affected pre-adult survival rates in 93% of extracted populations drawn from divergent animal taxa and environments. Second, these effects tended to be deleterious (thereby fitting the definition of maternal-effect senescence) across all broadly defined animal groups, with the conspicuous exception of birds from agricultural and wild populations. Maternal age trajectories tended towards concavity in all groups, indicating that rates of senescence intensified in old mothers in populations that senesce, and rates of improvement diminished in late life in those populations that do not senesce.

The general trends observed here are anticipated by recent evolutionary theory. Moorad and Nussey [31] integrated *Indirect Genetic Effects* (IGEs) into evolutionary demographic models of phenotypic selection with the aim to predict how maternal age should evolve to affect pre-adult survival. IGEs are a quantitative genetic concept developed by animal breeders [57,58] before gaining much attention from evolutionary geneticists interested in social evolution [59–61]. This begins with the conventional perspective that individual phenotypes (e.g., pre-adult survival) are affected by their own genes (*Direct Genetic Effects*, or DGEs) and the environment that they experience. However, it also recognises that one's environment can be affected by influences from social partners (e.g., mothers), and the social environment that produces the phenotype can evolve by natural selection to the degree that these influences are genetic (IGEs).

309 Phenotypes evolve as individuals' genetically-determined environments change by
310 natural selection.

311 Most evolutionary genetic models of senescence assume implicitly that DGEs
312 represent the only route towards evolutionary change [26,28,62], but Moorad and
313 Nussey [31] modified one such model [30] by assuming that mothers contributed IGEs
314 that were independent and identically distributed across all ages. They found that as
315 maternal age increases, selection to remove deleterious age-specific IGEs must eventually
316 diminish, and it follows that the pre-adult survival should evolve such that it deteriorates
317 as mothers get older. Furthermore, they showed that certain demographic conditions
318 exist that allow pre-adult survival to evolve to *increase* with maternal age at early ages
319 before reversing and evolving senescence at late ages, but they emphasised that all
320 models eventually lead to accelerated rates of senescence with increasing age. This
321 prediction is consistent with the pervasive negative quadratic relationships observed
322 here, although it must be noted that Moorad and Nussey evaluated death rates on a
323 different scale than that used here in our probit models, and we cannot state with
324 confidence that a negative quadratic relationship on one scale reliably predicts the same
325 on the other. This issue can be explored explicitly in follow-up studies that can focus more
326 directly on testing evolutionary theory (see below).

327 One goal of our study was to describe the diversity of maternal ageing. In particular,
328 we were interested in the role of phylogeny. A secondary focus was to characterize how
329 human influence upon populations (e.g., laboratory vs wild populations) might affect age
330 effects. However, it was clear from our review of the relevant literature that phylogeny
331 and environment are too closely aligned to make broad conclusions regarding
332 independent effects of both factors. Consequently, we were largely forced to consider our
333 primary focus, phylogeny, but it should be understood that our suggested causal

334 inferences regarding these should be considered preliminary until enough studies that
335 disrupt the association between phylogeny and environment are published to support
336 more focused reviews. Such efforts are being made to study actuarial senescence, such as
337 in wild insect populations [63–68], and we encourage more such work to explore
338 maternal-age effects.

339 We found a reasonable number and diversity of studies (species number > 5 and
340 population replicates > 10) in laboratory invertebrates, wild birds, wild mammals, and
341 humans. Whilst maternal age in older than average individuals was clearly deleterious
342 when averaged over all groups, two taxonomic groups stood out as clearly different. Wild
343 birds were the only reasonably populated group that exhibited *positive* effects of
344 maternal age in the old. Why birds should be so different in this respect is an outstanding
345 question that requires further study, but we might speculate on possible causes.

346 Pre-adult birds are frequently cared for by individuals other than their mothers. The
347 most common source of additional care is the father [69,70]. It may be that maternal-
348 effect senescence exists in birds, but these deleterious effects are masked by sources of
349 allocare. Unless the ages of other care-givers are perfectly correlated with those of the
350 mothers, we can expect that offspring of old mothers will receive care from young social
351 partners and vice versa. This will obscure the effects of maternal age (an effect of a
352 reversion-to-the-mean). A total of 17 of the 20 (85%) of the surveyed wild bird species
353 are known to exhibit some form of biparental care; this approximates the prevalence of
354 80-90% across all bird species [70]. Biparental care is comparatively rare in mammals
355 [71] and invertebrates [72]. Only four of eleven surveyed wild mammal species provide
356 this form of care [71], and no surveyed invertebrate species is known to demonstrate
357 biparental care [72]. Viewed across the non-human groups studied here (invertebrates,
358 wild mammals, and wild birds), the frequency of biparental care appears to counter-

359 indicate the degree of maternal-effect senescence, but this pattern is only suggestive;
360 more studies are needed from wild mammal and invertebrate species that exhibit
361 biparental care and from bird species that do not. Interestingly, one relevant and highly-
362 replicated laboratory study on an insect with biparental care, *Nicrophorus vespilloides*,
363 found no effects of maternal age on pre-adult survival [19]. Furthermore, we note that
364 even when biparental care is absent, paternal age can still have an effect on offspring
365 outcomes through other mechanisms, such as sperm quality. For example, increased
366 paternal age in the long-lived houbara bustard (*Chlamydotis undulata*) causes both a
367 decline in hatching success and rate of pre-adult development [73,74]. Studies should
368 account for variation in paternal age either by reducing or eliminating it via the
369 experimental design (e.g., [19]) or accounting for it statistically by model fitting (e.g.,
370 [75]). Observations from human populations do not support biparental care as a primary
371 cause for the wild bird results. Father and grandmothers can contribute meaningfully to
372 the performance of infants and children, but humans appear to have strong signatures of
373 maternal senescence when compared to other vertebrates, the majority of which provide
374 only uniparental care. Finally, it must be noted that this suggested mechanism can only
375 serve to reduce the apparent magnitude of maternal-age effects; it cannot reverse their
376 direction.

377 In terms of the magnitude of effects, Lepidopterans (moths and butterflies) were
378 clearly the most disparate group with extremely deleterious average effects in the old, an
379 order of magnitude greater than the other groups combined. Even when compared to
380 non-Lepidopteran invertebrates (which still exhibited stronger effects than vertebrates),
381 these rates were seven-fold greater. Variation in the nature of maternal care might
382 account for these differences. None of the studied invertebrate species delivered post-
383 natal care, yet many vertebrate studies focused upon juvenile periods coincident to post-

natal care. If pre-natal maternal-effect senescence was stronger than post-natal senescence, then senescence in invertebrate studies would tend to be stronger. One way that this might happen is if increased age provides some mitigating benefit to the pre-adult. Learning, for example, is believed to cause increased fledgling rates with increased maternal age in seabirds [76,77], and it is difficult to imagine how increased experience can serve to improve pre-natal condition to the same degree. However, this suggestion does not explain why Lepidopterans age differently from other invertebrates. Pre- and post-natal maternal-effect senescence has been measured independently in very few studies, including seabirds [78] and burying beetles [19]; more such studies made over a diversity of species are necessary to assess general patterns of pre- vs post-natal maternal-effect senescence.

Another striking life history difference between studied vertebrate and invertebrate species is that the duration of reproductive lifespan of the former is longer than that of the latter, even when accounting for the vast differences in generation time. One way to quantify this is to take the square-root of the variance in standardized maternal age at birth, or $\sqrt{\sum_x \left(\frac{f(x)}{\bar{T}} - 1\right)^2}$, where $f(x)$ is the fraction of new offspring attributed to mothers of that age, and $\bar{T}$ is the mean of that distribution (see Materials and Methods). This provides a dimensionless comparative metric of the dispersion of maternal age (sigma). The medians of sigma for vertebrates and invertebrates are 0.347 and 0.178, respectively. Evolutionary theory anticipates that maternal senescence should evolve to be faster when sigma is small, although that prediction has not been made previously. Moorad and Nussey [31] showed that in stable age-structure populations, the strength of selection acting on an IGE produced by a mother of some age that acts on pre-adult survival is proportional to the probability $f(x)$, the same distribution that defines

generation time T (the mean) and the standard deviation about the standardized mean (sigma). It must be the case that when sigma is small, selection for IGEs declines more precipitously after T than when sigma is large. This leads to the prediction that maternal-age effects in the old are positively associated with this measure (senescence decreases as sigma grows). This might explain why Lepidopterans (median sigma = 0.069), which are usually considered to have reproductive lifespans that are so abbreviated as to make them nearly semelparous [79], senesce faster than other invertebrates (median sigma = 0.282). Further evidence for positive associations between sigma and rates of ageing come from within-group Spearman rank correlations: Lepidopterans (0.538, n = 27); Non-Lepidopteran Invertebrates (0.177, n = 79); Wild Birds (0.0325, n = 37); and Wild Mammals (0.112, n = 12). We note, however, that Lepidopterans are the only group with a correlation estimate that reaches significance (P = 0.004).

Finally, we note that the evolutionary theory [31] emphasized that selection for age-specific maternal IGEs for pre-adult survival follows entirely from mean vital rates. As vital rates for many species are now available (e.g. [80,81]), selection for maternal-age effects can be estimated directly over a diverse collection of populations. An obvious and tractable question that remains to be investigated is whether variation in selection explains the diversity of ageing rates within and among the groups identified here. No formal attempt has been made to reconcile patterns of selection with observations of actuarial or reproductive senescence on such a broad scale. However, this could be done in conjunction with the aforementioned analysis to evaluate which manifestations of ageing (maternal, actuarial, or reproductive) adhere closest to predictions made from evolutionary theory.

While we make no specific suggestions for why these might generate the particular patterns of diversity observed here, we believe that two other factors deserve to be

mentioned. First, the manner by which natural selection affects an evolutionary change is sensitive to the genetic architecture underlying the trait [82]. Quantitative genetic approaches can be applied to characterize genetic architecture in wild populations [83–86], but we are aware of no attempts to do this for age-specific maternal IGEs. Second, most relevant ageing studies measure cohort-level changes in the effects of maternal age. These reflect a combination of within- and among-individual changes. Evolutionary models (including [26,31]) focus on the former, which makes testing predictions using cohort-level measures risky. The most obvious source of among-individual change is selective disappearance [2,8,87,88]. Studies of maternal-effect senescence can quantify these effects, but this is rare. Among studies that do measure it, there does appear to be variation in its importance [19,51,89,90]. Evaluating the effects of selective disappearance is relatively straightforward (see cited examples), and all ageing studies should attempt to do so.

Previous studies have documented and attempted to explain among-species diversity in rates of actuarial and reproductive senescence [3,4,91]. This work extends this effort to another manifestation of ageing. Consistent with these earlier studies, we find obvious among-species variation in rates of ageing with clear evidence for underlying structure involving phylogeny. Whilst the causes for this structure are still unclear, we are encouraged that general patterns appear to be consistent with predictions from evolutionary theory, and we are hopeful that finer-scaled tests of this theory will shed light on the causes of variation in rates of maternal ageing. Future experimental and observational studies on maternal-effect senescence will improve our ability to explain this variation, especially when they focus upon understudied taxa (e.g., fish, reptiles and amphibians), wild populations of invertebrates, and species with life histories that appear unusual for their taxonomic groupings (such as mammals or insects that exhibit

458 paternal or cooperative care). Taxonomic gaps amongst wild populations likely reflect a
459 general preference amongst ecologists to invest in long-term studies of birds and
460 mammals rather than any biological feature of these species that might make them more
461 amenable to the study of ageing. Finally, it would be interesting to assess maternal ageing
462 in species that lack evidence for actuarial senescence [see 4] (these species did not appear
463 in our search for data amenable to our analyses), particularly as evolutionary theory
464 predicts a link between adult ageing rates and the evolution of maternal senescence [31].
465
466 Authors' Contributions
467 EIC and JM conceived the ideas, designed the methodology, analysed the data and wrote
468 the manuscript. EIC collected the data. Both gave final approval for publication.
469
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478
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Figure Legends

Fig. 1 Maternal-age effects in old individuals arranged by phylogenetic relationship. Effect sizes and SEs (on the probit scale) were averaged within species. Three replicates were removed prior to averaging as their SEs made visualisation impossible (*Centropages typicus*, -78.53 ± 8991.38; *Psuedaletia sequax*, -159.74 ± 6298.32; and *Ovis aries*, 14.23 ± 2474.36). Two species indicated by * were not included here because their estimates would not fit (see Table S3b for these). Colours/line-type indicate environment: wild (blue/two-dashes), laboratory (red/dot-dash), semi-captive (purple/long-dash), agricultural (black/solid), and humans (orange/dotted). Error bars around the estimate represent 95% confidence intervals.